IN THE CLAIMS

Please amend the claims as follows:

D'

26. (amended) The method of Claim 24 ~~wherein the polycystin-1 protein is~~ expressed to a higher level as compared to endogenous polycystin-1 protein.

REMARKS

Claims 21-37 are currently pending. Claim 21-23 are rejected under 35 U.S.C. §103. Claim 26 is rejected under 35 U.S.C. §112, second paragraph. Claim 26 has been amended to more particularly point out and distinctly claim the invention. No new matter is introduced by the amended claim and the claim is fully supported by the instant specification. For reasons set forth in detail below, Applicants request that the rejections be withdrawn and the claims be allowed to issued.

1. Claim 26 as Amended is Definite

Claims 26 is rejected under 35 U.S.C. §112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The Examiner alleges that the phrase "over expressed" in Claim 1 is relative. To more particularly point out and distinctly claim the subject matter which applicant regards as the invention, Applicants have amended Claim 26 to indicate that the polycystin-1 protein is expressed to a higher level as compared to endogenous polycystin-1 protein.

In view of the foregoing remarks and amendment to claim 26, the rejection under 35 U.S.C. §112, second paragraph, should be withdrawn.

2. The Claims Are Not Obvious

Claims 21-23 are rejected under 35 U.S.C.§103 as being unpatentable over Wilson et al., (1996) in view of Van Adelsberg. The Examiner alleges that Wilson teaches the correlation between PKD-1 content and degree of adherence to type 1 collagen and Van Adelsberg teaches peptide inhibitors derived from PKD repeats of polycystin-1. Thus, accoding to the Examiner, it would have been obvious to one of skill in the art at the time the invention was made to measure adherence of a derivative of polycystin-1 expressing cells to collagen type-1 in the presence of the inhibitory peptides derived from the PKD repeats of polycystin-1 as taught by Van Adlesberg, with a reasonable expectation of success. The Examiner maintains that one of skill in the art at the time the invention was made would have been motivated to make this modification to determine if type 1 collagen is a ligand for polycystin-1.

A finding of obviousness under §103 requires a determination of the scope and content of the prior art, the level of ordinary skill in the art, the difference between the claimed subject matter and the prior art, and whether the differences are such that the subject matter as a whole would have been obvious to one of ordinary skill in the art at the time the invention was made. *Graham v. Deere*, 383 US1 (1996). The relevant inquiry is whether the prior art suggests the invention and whether the prior art provides on e of ordinary skill in the art with a reasonable expectation of success. In re O'Farrell, 853 F.2d 894, (Fed. Cir. 1988).

In the present instance the relevant inquiry is whether Adelsberg in combination with Wilson would render the presently pending claims obvious. Clearly, the answer to that question is no.

Claims 21-23 are directed to screening assays designed for identifying modulators of PKD-1 activity. Such screening assays are based on Applicant's discovery that cells expressing PKD-1 protein display increased adherence to type 1 collagen coated surfaces.

Applicants assert that van Adelsburg fails to disclose a single screening assay designed to identify novel compounds capable of modulating PKD-1 activity. van Adelsburg merely suggest that peptides derived from PKD repeats may be used to ***study*** branching morphogenesis in developing kidney. Specifically, as stated by van Adelsburg, "if polycystin-1 is a receptor, then fragments of its ligand binding domain should act as competitive inhibitors by binding to candidate polycystin ligands." (see, van Adelsberg p.302, col.1, last paragraph). Thus, van Adelsberg is merely interested in studying the process of branching morphogensis, not development of screening assays for identifying PKD-1 modulating compounds. Furthermore, the procedure used by van Adelsburg involves the use of organ cultures. Specifically, kidney rudiments are cultured in the presence of PKD repeats for four days followed by counting of glomeruli in whole mounts of cultured kidney rudiments. Such a procedure would be consider much to laborious and time consuming to provide a practical screening assay. In addition, the deficiency in van Adelsburg is not supplied by the Wilson reference, which also fails to disclose, or suggest, screening methods useful for identification of compounds capable of modulating PKD-2 activity.

In light of the above, Applicants respectfully request that the rejections under 35 U.S.C. §103(a) be withdrawn.